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Epigenetics Research: Open Access

Short Communication

Short Note on Gene Silencing in Bacteria using CRISPR Interference

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DESCRIPTION

Negative feedback mechanisms like gene silencing control gene expression to determine a cell's and regulate metabolism and gene expression throughout an organism's life. Post-Transcriptional Gene Silencing (PTGS) and Transcriptional Gene Silencing (TGS) are the two methods by which genes in plants can be silenced. In nature, RNA silencing is common. There are a variety of mechanisms and cellular functions described for the involved transcripts, which are frequently referred to as non-coding regulatory RNAs or antisense RNA. These normal instances of RNA quieting give significant data about how manufactured RNA methodologies can be best evolved [1].

Trans and cis-antisense sequences in bacteria are examples of reversibly repressing translation. An intramolecular antisense-sense fold can be formed by adjacent cis-antisense sequences in the regulatory regions of a single mRNA. The folded structure prevents initiation because it conceals or conceals the Ribosome-Binding Site (RBS). Although this arrangement is less obvious as an antisense control mechanism, it was first described in 1985 and has since been linked to the regulation of numerous genes [2]. Additionally, it is now clear that transcription from the complementary strand at the same locus can result in the formation of cis-antisense sequences, which occur as frequently in simple unicellular bacteria as they do in higher organisms. Trans-acting regulatory RNAs, in which the antisense RNA is transcribed from a distant locus, make up many natural antisense sequences. Due to the anticipated nature of antisense, the discovery of such trans-encoded RNAs may be favoured however; it appears likely that this is a typical natural antisense mechanism. Trans-encoded antisense sequences typically function in prokaryotes by binding to the mRNA's start codon region. OxyS RNA, on the other hand, prevents the translation of two target genes-fhlA and rpoS while MicF RNA represses the translation of the outer membrane protein gene *ompF*. Bacteria can silence RNA in a variety of ways [3-5].

There are several ways that antisense sequences that hybridize with messenger RNA can prevent the expression of target genes; analyzed in depth in Translation repression may be the most straightforward mechanism. In bacteria, where the ribosome binds to mRNA at the well-known RBS, this is straightforward to imagine. When an antisense sequence is positioned at the RBS to stop this initiation process, the unused transcript decays and the messenger RNA is revealed. Natural antisense transcripts have been shown to play additional confirmed functional roles in transcription termination, co-degradation, transcriptional interference, and enhanced stability of their respective target transcripts. In addition, the RNA-mediated destruction of phage transcripts is how the recently discovered Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) mechanism confers acquired resistance against bacteriophage. As a result, a wide range of potential applications for synthetic RNA-silencing tools can be found in the natural antisense mechanisms and roles they play.

The first natural antisense mechanisms that were discovered were thought to be unique aspects of gene expression control and were associated with accessory elements found in bacteria. It is now abundantly clear that antisense mechanisms play a role in both adaptive responses and fundamental cellular processes. As a result, RNA silencing is a common and significant method of controlling posttranscriptional genes [6-8].

It has been said that the structure and function of RNA are highly evolvable, and interactions between RNAs can alter biological processes in subtle and profound ways. These characteristics are advantageous to bacteria, and a remarkable array of RNA-level regulatory processes has developed. Antisense translation repression is one of these that are best understood [9]. Rapid expression switching in response to cellular and environmental signals is a key feature of translation repression, which allows the mRNA to remain intact during periods of repression. It seems reasonable for cells to constitutively transcribe antibiotic resistance genes and then control expression at the translation level because exposure to antibiotics requires a rapid response to ensure cell survival. *E. coli's* chloramphenicol and erythromycin resistance genes contain antisense sequences encoded by cis. Antisense sequences within a brief open reading frame just upstream of the start codon appear to provide rapid expression control for the chloramphenicol and erythromycin resistance genes. Additionally, RNA-silencing appears to permit operon-specific

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Received: 22-May-2023, Manuscript No. EROA-23-25346; **Editor assigned:** 24-May-2023, PreQC No. EROA-23-25346 (PQ); **Reviewed:** 07-Jun-2023, QC No. EROA-23-25346; **Revised:** 14-Jun-2023, Manuscript No. EROA-23-25346 (R); **Published:** 21-Jun-2023, DOI: 10.35248/EROA.23.5.137

Citation: Bohman E (2023) Short Note on Gene Silencing in Bacteria using CRISPR Interference. J Epigenetics Res. 5:137.

gene control. For instance, Spot 42 RNA repression of galK at an internal location within the galETKM operon transcript effectively disassociates expression from a normally coordinated expression system. RNA silencing is also a part of the bacterial CRISPR defense mechanism against viral infection. As a result, natural RNA silencing is involved in a wide range of phenotypes, including antibiotic and phage resistance, and involving RNA-level gene control clearly benefits the cell [10,11].

CONCLUSION

Implementation of *S. aureus* target array technology for a wide range of pathogenic bacteria and the determination of target stringency for known essential genes in a number of gram-negative and positive species, as well as the correlation of data to known antibacterial targets. In order to take advantage of the potential of oligo-nucleobase RNA silencers like PNAs and PMOs, it may be necessary to develop second-generation carrier strategies that may involve non-biological solutions. An examination of the efficacy of PNA against *E. coli* leaky mutants suggests that improved delivery could significantly enhance the drug's efficacy. Additionally, a variety of carrier molecules may aid in the delivery of specific species or infection sites. Polypharmacological oligo-nucleobase RNA silencers will also become more effective against bacteria. The clinical evaluation of RNA silencers could be accelerated as a result of these advancements, potentially leading to a new class of antimicrobial compounds with distinctive design characteristics.

REFERENCES

1. Agrawal N, Dasaradhi PV, Mohmmed A, Malhotra P, Bhatnagar RK, Mukherjee SK. RNA interference: Biology, mechanism, and applications. Microbiol Mol Biol Rev. 2003;67(4):657-685.
2. Brouns SJ, Jore MM, Lundgren M, Westra ER, Slijkhuis RJ, Snijders AP, et al. Small CRISPR RNAs guide antiviral defense in prokaryotes. Science. 2008;321(5891):960-964.
3. Caro F, Place NM, Mekalanos JJ. Analysis of lipoprotein transport depletion in Vibrio cholerae using CRISPRi. Proc Natl Acad Sci. 2019;116(34):17013-1722.
4. Karvelis T, Gasiunas G, Miksys A, Barrangou R, Horvath P, Siksnys V. crRNA and tracrRNA guide Cas9-mediated DNA interference in Streptococcus thermophilus. RNA Biol. 2013;10(5): 841-851.
5. Li XT, Jun Y, Erickstad MJ, Brown SD, Parks A, Court DL, et al. tCRISPRi: Tunable and reversible, one-step control of gene expression. Sci Rep. 2016;6(1):39076.
6. Man S, Cheng R, Miao C, Gong Q, Gu Y, Lu X, et al. Artificial trans-encoded small non-coding RNAs specifically silence the selected gene expression in bacteria. Nucleic Acids Res. 2011;39(8):e50.
7. Jansen R, Embden JD, Gaastra W, Schouls LM. Identification of genes that are associated with DNA repeats in prokaryotes. Mol Microbiol. 2002;43(6):1565-1575.
8. Beisel CL, Storz G. Base pairing small RNAs and their roles in global regulatory networks. FEMS Microbiol Rev. 2010;34(5):866-882.
9. Wang C, Cao Y, Wang Y, Sun L, Song H. Enhancing surfactin production by using systematic CRISPRi repression to screen amino acid biosynthesis genes in *Bacillus subtilis*. Microb Cell Fact. 2019;18:1-3.
10. Hou Y, Ma W. Natural host-induced gene silencing offers new opportunities to engineer disease resistance. Trends Microbiol. 2020;28(2):109-117.
11. Depardieu F, Bikard D. Gene silencing with CRISPRi in bacteria and optimization of dCas9 expression levels. Methods. 2020;172:61-75.